

SHORT COMMUNICATION**Case report****Bilateral orbital hemorrhage induced by labor**S. ORUÇ¹, E. CUMHUR ŞENER², A. AKMAN³, A. ŞEFİK SANAÇ²¹Department of Ophthalmology, Adnan Menderes University Medical School, Aydın²Department of Ophthalmology, Hacettepe University Medical School, Ankara³Department of Ophthalmology, Başkent University Medical School, Ankara - Turkey

We describe a woman in whom bilateral orbital hemorrhage occurred during labor. She developed sudden proptosis and complete loss of vision bilaterally. After a stillbirth, she underwent total hysterectomy because of atonic uterus and postpartum hemorrhage. The location of the hematomas was confirmed by magnetic resonance imaging. Clinical resolution occurred in one month but both eyes remained blind. Fundoscopy revealed bilateral atrophy of the optic discs. (Eur J Ophthalmol 2001; 11: 77-9)

KEY WORDS. *Orbital hemorrhage, Labor, Blindness, Valsalva maneuver, Optic neuropathy*

Accepted: January 31, 2000

INTRODUCTION

Common causes of orbital hemorrhage include trauma, surgery, vascular anomalies, tumors, blood dyscrasias, and Valsalva maneuver. It has rarely been reported during labor (1-6). Anterior ischemic optic neuropathy is rare in the young, but it has been described in association with a number of conditions such as collagen vascular diseases, hypertension, migraine, and blood loss (7).

We would like to present a 23-year-old woman who suffered bilateral orbital hemorrhage during labor and developed complete loss of vision bilaterally.

Case report

Four days before presentation at Hacettepe University Hospital, a 23-year-old woman had entered spontaneous term labor. This was the patient's first pregnancy and preeclampsia had developed. After delivery of the baby's head the labour had slowed down, proptosis of both eyes had developed and the patient had lost her vision bilaterally. The infant was stillborn, and total hysterectomy was performed because of atonic uterus and postpartum hemorrhage, and a blood transfusion was given.

She was referred to Hacettepe University Hospital. On presentation, her visual acuity was no light perception OU. Motility was minimally restricted in all fields of gaze and exophthalmometry measurements were 21 mm at both eyes, showing symmetrical bilateral proptosis. The pupils were both the same size and non-reactive to light. Slit-lamp biomicroscopy and tonometry results were unremarkable, but dilated fundus examination showed blurring of the optic disc margins bilaterally associated with flame-shaped hemorrhages in the area of the optic disc on the left eye. Blood indexes and clotting parameters were normal.

Magnetic resonance (MR) imaging demonstrated bilateral intraorbital, extraconally located hematomas. Dilated vascular structures were identified in the left orbit medial to the globe, suggesting a vascular malformation. The cavernous sinuses were unremarkable. The superior ophthalmic veins were symmetrical with normal caliber bilaterally. There was no destruction of the bony structures of the orbit (Figs. 1, 2). MR imaging of the head gave results within normal limits.

Management was limited to observation. The proptosis resolved gradually over the next four weeks with no residual deficits. Fundoscopy revealed clearance of the flame-shaped hemorrhages and bilateral atro-

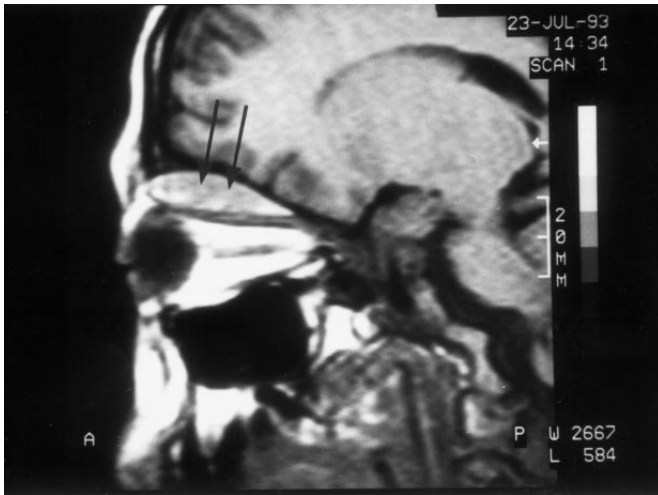


Fig. 1 - T1 weighted parasagittal image (TR/TE = 520/20 msec) shows an extraconal mass hypertense to muscle above the superior rectus muscle, representing the orbital hematoma in the right orbit (arrows).

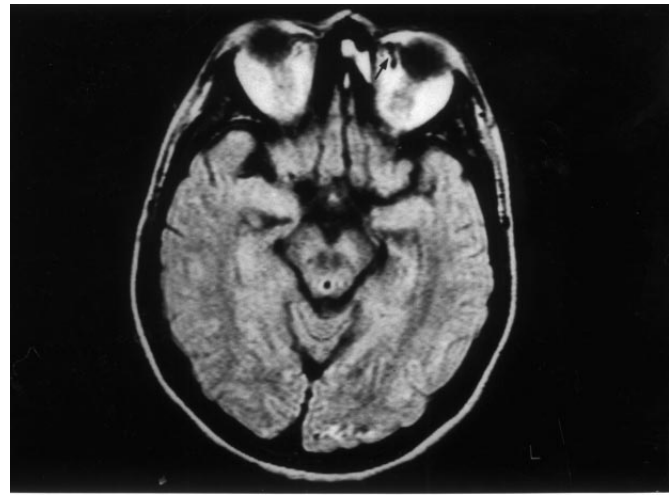


Fig. 2 - Proton density axial image (TR/TE=2001/20 msec) shows a dilated vascular structure medial to the globe on the left (arrow), suggesting a vascular malformation.

phy of optic discs. No light perception was present bilaterally. The patient refused to have a repeat MR imaging after the orbital hemorrhage resolved in order to confirm orbital vascular malformation.

DISCUSSION

Spontaneous orbital hemorrhage may be associated with cavernous hemangioma, lymphangioma, orbital venous anomalies, idiopathic inflammatory pseudotumor, hypertension, hemophilia, blood dyscrasias, leukemia, renal disease, and scurvy. Hemorrhage associated with Valsalva maneuver has been reported rarely during labor, or weight lifting (1-6). Straining associated with the Valsalva maneuver increases intra-abdominal and intrathoracic pressures. The resulting increase in jugular venous pressure is transmitted to the orbit by valveless veins (3, 5). Venous distention may then lead to hemorrhage. Orbital hematoma can result in optic neuropathy by the compression from the hematoma and the direct effect of blood waste product on the optic nerve. It is probable that in our patient the orbital hematoma was caused by the Valsalva maneuver which raised the intravascular pressure, that was then transmitted to the orbital veins, which lack valves. Bilateral vascular structures suggesting vascular abnormalities, and hypertension, might be predisposing factors in this case. As the patient refused to repeat MR imag-

ing after the orbital hemorrhage resolved, we could not confirm orbital vascular malformation.

Spontaneous orbital hemorrhage during labor has, to our knowledge, been reported three times in patients who had normal or slightly decreased visual acuity (1,5,6). Our patient is the first reported case of bilateral orbital hemorrhage during labor resulting in bilateral loss of vision. The patient was preeclamptic and underwent hysterectomy after a stillbirth because of atonic uterus and postpartum hemorrhage. Several case reports of ischemic optic neuropathy resulting from hemorrhage have been reported; approximately 30% are the result of uterine hemorrhage. Visual symptoms ranged from blurred vision to total vision loss (8, 9). Johnston et al (10) suggested that the locus of optic nerve infarction after a hypotensive episode might depend on preexisting risk factors. Acute systemic hypotension by itself is unlikely to cause optic nerve infarction in young patients. Possibly in our patient a hypotensive episode secondary to uterine hemorrhage may cause a further reduction in perfusion to the optic nerve which is already vulnerable because of the orbital hemorrhage. It is tempting to accept the suggestion that both compression and ischemia of the optic nerve head are the reason for blindness in our patient.

The retrobulbar hematoma requires special attention because potential compression of the optic nerve may impair vision or cause blindness. We recommend

observation as the initial management in patients without elevated intraocular pressure or optic neuropathy. In case of visual loss immediate surgical drainage is indicated. This patient was not considered for surgery because no light perception was present on admission and four days had passed before she reached our hospital. Immediate, adequate blood transfusion may prevent or limit the severity of vision loss in ischemic optic neuropathy secondary to blood loss.

Any complaint of visual loss during labor should be investigated with visual acuity and confrontational vi-

sual field examinations. If these are abnormal, full evaluation and treatment must be ensured in a very short time, otherwise partial or total loss of vision can be the outcome, as in this case.

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